Fig. 1.118 Characteristic impairment and retardation of nasal growth in identical twins at the age of 14 years.
The boy on the right **(b)** suffered from nasal trauma with subsequent septal infection (small abscess) at the age of 8 years. The abscess was drained but the defect was not repaired. When we compare him with his twin brother on the left **(a)** 6 years later, we notice a typical retardation of nasal growth.
a, b In the boy on the right **(b)** the height of the nasal pyramid is much less than in his twin brother.
c, d In the boy on the right **(d)** the length and prominence of the external nose are limited; the bony and cartilaginous pyramid is broader and saddling; the lobule is low and wide; the tip is underprojected; the columella is short and somewhat retracted; the nasolabial angle is increased.
e, f In the boy on the right **(f)** the length and prominence of the external nose are limited; the bony and cartilaginous pyramid is broader and saddling; the lobule is low and wide; the tip is underprojected; the columella is short and somewhat retracted; the nasolabial angle is increased.
g, h In the boy on the right **(h)** the lobule is severely underprojected and wide; the columella is short and somewhat retracted.
From Huizing 1966.

Huizing (1966), Pirsig (1992), and Grymer (1997). The earlier (and the more destructive) the trauma, the more severe the growth disturbance and the ultimate nasal pathology.

Childhood Septal Abscess

A septal abscess in childhood poses the greatest threat to normal nasal growth. Unless treated by septal reconstruction as soon as possible, it will lead to severe disturbance of the outgrowth of the septum, pyramid, and midface. A characteristic example of the growth disturbance caused by trauma and septal abscess in childhood is presented in ▶ Fig. 1.118.

1.4 Surgical Physiology

Six different nasal functions may be recognized, in their phylogenetic order:
1. Olfaction
2. Respiration
3. Climatization (heating and humidification of inspired air)
4. Defense of the respiratory tract
5. Speech production
6. Facial expression and beauty

When, during the process of evolution, air became the medium of life instead of water, the nose developed. This required the development of a special tract with new

provisions: the respiratory tract. At its entrance, a chemical sense developed—the olfactory organ—that differs from that within the mouth of sea animals. Moreover, an elaborate system to prepare the inhaled air in an optimal way for the lower respiratory tract developed, its functions including heating, humidification, and partial cleansing of particles. For that particular purpose, the nose consists of two parts: a right and a left. They work together but independently.

The human nose consists of an external and an internal nose. Only human beings and some types of ape have an external nose. From a physiological point of view, the external nose or nasal pyramid should first be considered as a regulator of airway resistance and airflow, as well as an organ of defense (function as an air filter). The reason that the external nose evolved in a plane in front of the face is not fully clear. It was probably an adaptation to changes in the conditions of life. The differences in the shape of the external nose between various human races suggest that adaptation to climatic conditions played a major role. Anthropological studies have revealed a close relationship between morphological features of the human nasal skeleton and the geographical climate, resulting in variations in nasal morphology. This connection emphasizes the fact that, in the context of evolution, adequate respiratory function of the nose is essential for ideal pulmonary gas exchange.

The internal nose is the actual and original nasal organ. The basic tasks of olfaction, climatization of inspired air, and defense take place within the three different functional segments of the internal nose (▶ Fig. 1.119). The anterior segment is the inflow of the nose. It includes the vestibulum, isthmus, and the anterior part of the nasal cavity. Physically, it functions as a flow manifold, nozzle, and diffuser, providing maximum contact between air and mucosa. The anterior segment, including the nasal valve area, is responsible for alteration of the nasal airflow. The airflow pattern is disrupted, spreading the air over the mucosa of the adjoining turbinates to allow optimal respiratory function within the middle functional segment.

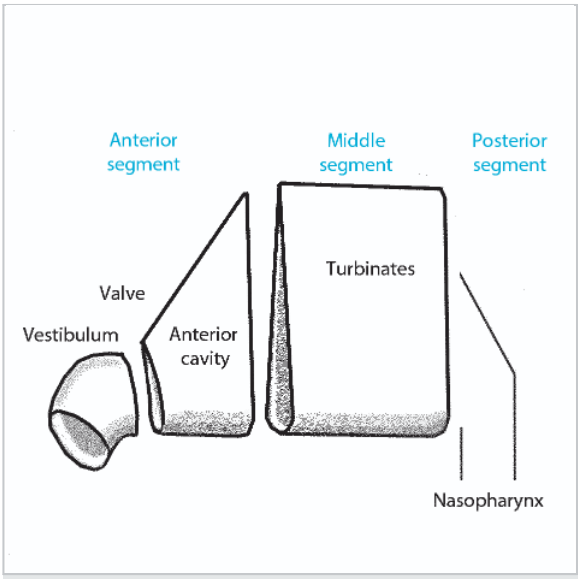

Fig. 1.119 Schematic representation of the functional anatomy of the human nasal organ.

1.4.1 Olfaction

Olfaction serves a variety of purposes. It helps locate food as well as water, and find a partner of the other sex. It also warns against the approach of enemies and the danger of environmental gases. The sense of smell is fully mature at birth, indicating its utmost importance in the mother–child relation.

> "In surgery, function should prevail over form."

The nose is an organ with a number of highly important functions. When operating on this organ, the nasal surgeon may be pursuing various goals: curing nasal disease, reconstituting normal nasal function and form, or enhancing facial expression and beauty. In surgery, curing disease and improving nasal function must always prevail over enhancement of beauty, no matter how important and legitimate this objective might be in a given case. In nasal surgery, function should never be sacrificed for beauty.

The organ of smell is located in a narrow cleft of 1 to 2 mm in the anterior part of the superior nasal passage above the level of the middle turbinate, both on its lateral (ethmoidal) and medial (septal) wall (▶ Fig. 1.120). The surface area of the olfactory epithelium varies between individuals. In adults it usually covers about 200 to 400 mm^2. Even at birth it may be up to 500 mm^2. The olfactory epithelium contains about 20 million receptor cells. These cells are connected to unmyelinated fibers that, in small bundles, traverse minor openings in the anterior skull base (i.e., the lamina cribrosa of the ethmoid bone). In addition, tubular serous glands (Bowman's glands) are present.

The human olfactory epithelium is renewed every 60 days by apoptosis, dead cells being replaced by basal cells. The axons grow in a site-specific manner, meaning that the new axons grow to the places vacated by the old ones.

Odors must be either fat soluble or water soluble to be perceived. The total number of different odors that man is able to distinguish has been estimated at several million. The human species is nevertheless a very poor smeller compared to most mammals. For instance, the olfactory area in dogs is thirty times larger than in humans.

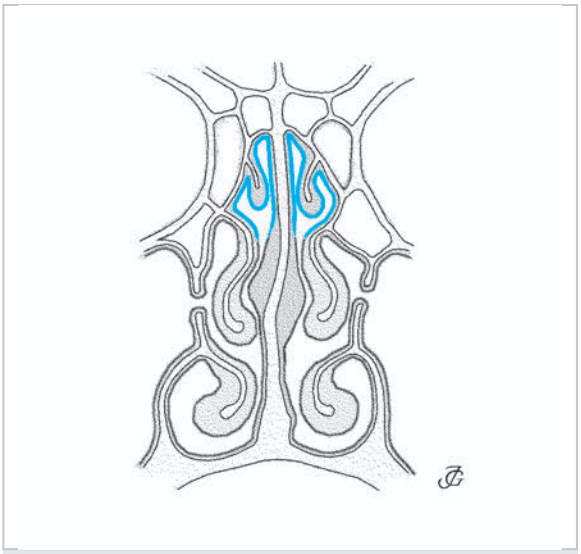

Fig. 1.120 Olfactory epithelium in the anterior part of the upper nasal passage or olfactory cleft, on the septum and the medial wall of the ethmoid bone.

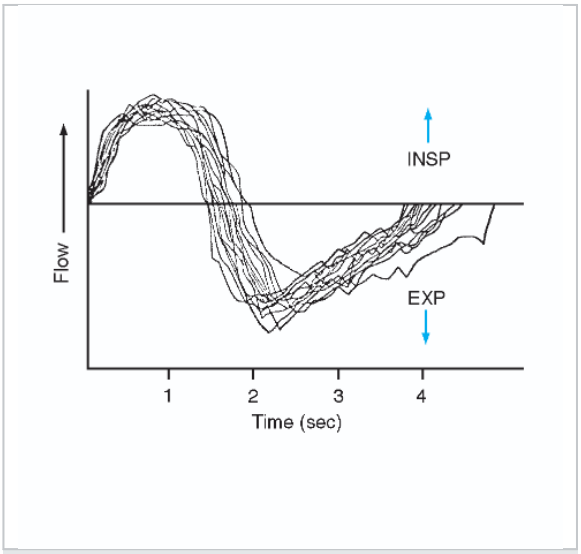

Fig. 1.121 Normal breathing cycle at rest. 1 = inspiratory phase; 2 = midcycle rest; 3 = expiratory phase; 4 = intercycle rest; INSP = inspiration; EXP = expiration. (From Cole 1982; data from Proctor and Hardy 1949.)

As only small amounts of air usually reach the olfactory region, in sensory analysis, the airflow is intensified by sniffing (the air is sucked in short bursts through the nose).

Loss of smell is a common complaint in ENT patients. Damage to the olfactory organ or nerve fibers (e.g., due to [viral] infections or anterior skull base fracture) may result in perceptive anosmia or hyposmia. Conductive anosmia or hyposmia may occur in cases when the inspired air fails to reach the otherwise intact sensory segment. Since the olfactory organ is located high within the nasal cavity and its access is narrow, conductive anosmia or hyposmia are common findings in rhinological practice.

Both types of disturbed olfaction may be differentiated by olfactory testing before and after decongestion of the mucosa caudal to the olfactory cleft.

1.4.2 Respiration

The major nasal function is breathing. The nose constitutes the first part of the respiratory tract and fulfills three major tasks within this system:

1. It provides the major part of respiratory resistance.
2. It facilitates close contact between air and mucosa due to changes in airflow patterns (increased turbulence and decreased velocity), allowing sufficient climatization.
3. It acts as the first line of defense for the protection of the lower respiratory tract.

Parameters of Breathing

The frequency of breathing in adults at rest is about 16 breaths per minute. According to ventilatory demands, it increases during exercise and decreases during sleep.

The volume of air inhaled in a single breath averages 500 mL. The total volume of air inhaled per day thus equals some 12,000 L. It is interesting that in humans, daily intake of air is approximately 12,000 L (= 12 m^3), whereas daily intake of water is about 2 kg, and that of food, 1 kg. All this air will be heated up to the body temperature of 37°C and humidified up to 100% relative humidity. The majority of air conditioning takes place within the nasal airways.

The velocity of the airstream depends on the force of respiration and the cross-sectional area and geometric shape of the nose at a given area. During normal inspiration, airflow velocity is 2 to 3 m/s within the nostril and 12 to 18 m/s within the nasal valve area (see also ▶ Fig. 1.127).

Respiratory Cycle

The normal breathing pattern consists of four main phases: inspiration, midcycle rest, expiration, intercycle rest (▶ Fig. 1.121). During breathing, graphical recording of the pressure changes at the level of the nostril, by means of a nozzle or by body plethysmography, may disclose abnormalities of the respiratory cycle. Pressure at the external nasal ostium equals 8 to 15 mm water at inspiration, and 2 to 4 mm less at expiration. It has been suggested (Cottle 1968, Heinberg and Kern 1974) that certain anomalies of the breathing pattern (e.g., a midcycle rest) might indicate imminent cardiopulmonary disease. However, a correlation between abnormalities of the respiratory cycle and cardiac disease has not been established yet.

Nasal Resistance and Its Effects

In nasal breathing, 50 to 60% of the total resistance of the respiratory tract is caused by the nose, in particular by the nasal valve area and the turbinates. During mouth breathing, the resistance of the upper airways decreases to less than 20% of total airway resistance. The nose creates a difference between the environmental air pressure and pressure within the lower respiratory tract. The major site of high nasal resistance is the nasal valve area, including the heads of the inferior and middle turbinates. The contribution to total nasal resistance by the nasal valve area, on the one hand, and the turbinates on the other, critically depends on individual nasal anatomy (e.g., ethnic factors, age, gender), the physiological state of the mucosa during the nasal cycle, and pathological abnormalities.

It is interesting to speculate for what purpose within phylogenetic development of the respiratory tract the nose has been added as a resistor of such magnitude. Two effects may be distinguished: pulmonary as well as cardiac effects, and local effects in the nose itself.

> "There is no real difference between structure and function: they are two sides of the same coin. If structure does not tell us anything about function it means we have not looked at it correctly."
> Szent-Gyorgyi

Pulmonary and Cardiac Effects of the Nasal Resistor

The most important pulmonary and cardiac effects of the nasal resistor are: a wider opening of the peripheral bronchioli and alveolar ventilation, allowing a more profitable gas exchange; and higher negative thoracic pressure resulting in better venous cardiac and pulmonary backflow. The nose also represents a source of nitric oxide, reaching the lower airways by inhalation and considered to be responsible for homeostasis of the bronchial tone and vasculature.

Local Effects in the Nose

Local effects result from the fact that the major nasal resistor, the nasal valve area, is located at the entrance of the nasal cavity. The nasal valve is a three-dimensional region, thus the term nasal valve area should be preferred. The nasal valve area is a triangular narrowing comprising the more or less rigid anterior septum and the soft tissue overlying the piriform aperture and nasal floor, the mobile caudal end of the triangular cartilage (valve), and the more or less swollen head of the inferior turbinate (▶ Fig. 1.122 and ▶ Fig. 1.123). It is remarkable that this area is the narrowest segment of the entire nasal cavity.

This anatomical–physiological narrowing at the entrance of the functional nasal segment causes a considerable increase in velocity of the inspired air (Bernoulli's law) from 2 to 3 m/s at the nostril to 12 to 18 m/s at the valve area. As a result, the outer layers of the laminar

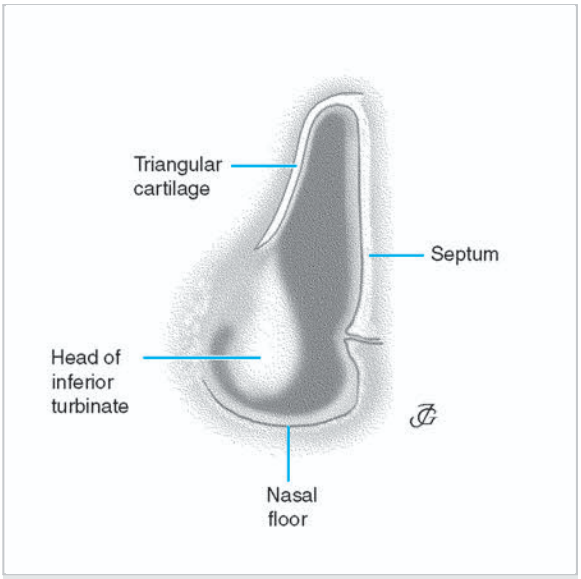

Fig. 1.122 The nasal valve area is the main nasal resistor. It consists of the mobile caudal margin of the triangular cartilage (the valve proper), the more or less swollen head of the inferior turbinate, and the semirigid septum and floor of the piriform aperture.

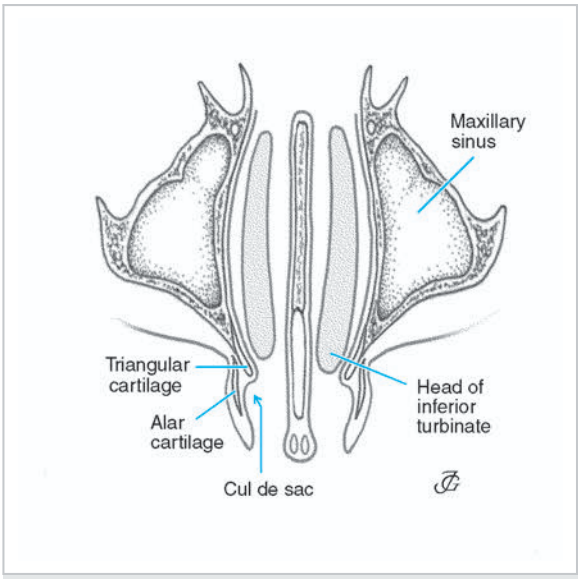

Fig. 1.123 The contribution of the inferior turbinate to the resistance of the valve area is variable. It depends on the degree of swelling of its head.

airstream become turbulent as Reynolds number is exceeded (see page 391). The valve area thus functions as an accelerator and consequently as a "diffuser" or "turbulizer" of the inspired air.

The anterior nasal segment, including the nasal valve area and the turbinates, plays a crucial role in air conditioning. The nasal valve area is responsible for alterations in nasal airflow: the airflow pattern is disrupted, spreading the air over the entire mucosa of the turbinates to allow heating and humidification of the inspired air. It acts as a diffuser, where turbulence increases and velocity decreases. When inhaled air passes the nasal valve area, the laminar airflow changes into a turbulent one, intensifying the contact between air and mucosa. Variations in the airflow pattern (velocity, flow, vortices, path lines) vary the degree of contact of the inhaled air with the surrounding mucosa. The kinetic energy of turbulent airflow allows maximal contact between the inhaled air and the mucosa. In laminar airflow, the direction of flow is parallel to the mucosal surface, with only the air film closest to the surface touching the nasal mucosa. In turbulent airflow, however, all of the air comes in contact with the mucosa due to mostly three-dimensional, random, and unsteady movements of the particles. There is a very close relationship between intranasal air conditioning and airflow patterns. Lower parts of the respiratory tract play a minor role in air conditioning (Lindemann 2006).

It is noteworthy that the increase in air temperature and humidity is higher within the short distance (about 1 cm) of the anterior segment than along the entire length of the middle turbinate (about 4 cm) (Keck 2000).

Resistors and Geometry of the Nose

When studying the dynamic behavior of the airstreams passing through the human nose, the physical laws postulated by Bernoulli (1738), Venturi (1788), Hagen-Poiseuille (ca. 1830), and Reynolds (1889) can be observed to hold. In connection with this topic, we refer to the section in the Appendix on physical laws governing airstreams. However, these laws of fluid physics apply to flow through a circular tube. The nasal cavity presents a completely different geometrical structure. As previously discussed, it consists of three anatomically and physiologically different segments. The anterior segment consists of three elements: an almost horizontal ovaloid opening (nostril), a funnel-shaped widening with various protrusions and pouches (vestibule), and a more or less triangular narrowing (valve area). The specific characteristics of the anterior segment are summarized in ▶ Table 1.2, ▶ Table 1.3, and ▶ Table 1.4.

The middle segment of the nasal cavity is a more or less trapezoid-shaped slit with very irregular lateral walls (▶ Table 1.5). The posterior segment or downstream area consists of the posterior end of the turbinates, the anterior wall of the sphenoidal sinuses, and the choanal opening (▶ Table 1.6).

Table 1.2 Geometrical data and resistors of the nostril (first element of the anterior nasal segment)

Nostril	
Position:	Horizontal
Shape:	Ovaloid
Cross-sectional area:	90 mm^2
Resistors:	Protruding ends of medial crura

Table 1.3 Geometrical data and resistors of the vestibule (second element of the anterior nasal segment)

Vestibule	
Position:	Oblique
Shape:	Funnel
Cross-sectional area from caudal to cranial:	90 → 120 → 60 mm^2
Resistors:	Protruding end of lateral crus Cul de sac

Table 1.4 Geometrical data and resistors of the valve area (third element of the anterior nasal segment and the transition between the anterior and middle nasal segment)

Valve area	
Position:	Oblique
Shape:	Triangular
Cross-sectional area:	50–70 mm^2
Resistors:	Valve (lower margin of triangular cartilage) Head of inferior turbinate Septum, floor of valve area

Table 1.5 Geometrical data and resistors of the middle nasal segment of the nasal cavity

Nasal cavity	
Shape:	Trapezoid-like slit
Cross-sectional area:	40/130/80 mm^2
Resistors:	Laterally: inferior, middle, and superior turbinates Medially: septal turbinates, septal folds

Table 1.6 Geometrical data and resistors of the posterior nasal segment

Choana	
Shape:	**Ovaloid**
Cross-sectional area:	80 mm^2
Resistors:	Tails of inferior and middle turbinates Anterior wall of sphenoidal sinus

All these segments, with their cross-sectional areas, specific geometry, and walls act as resistors and directors of airflow. They determine the course of the inspiratory and expiratory airstream and its velocity and turbulence behavior.

Pathway and Velocity of Inspiratory and Expiratory Airflow

The route taken by inspired and expired air has been the subject of numerous studies for more than a century and a variety of experimental and numerical models has been used for the analysis of airflow. The first investigators at the end of the 19th century thought that the pathway of both the inspiratory and expiratory airstream was through the inferior nasal passage. Later, experiments on cadaver specimens and other models demonstrated that the inspiratory airstream takes a higher, curved course, while the expiratory airstream follows the lower nasal passage (Paulsen 1882, Franken 1894, Goodale 1896, Courtade 1903, Mink 1920, Proetz 1951). Van Dishoeck (1936) demonstrated in model experiments that the course of the inspiratory airstream was influenced by the position of the nostril: the smaller the nasolabial angle, the higher the course. More recently, the inspiratory airstream has been further analyzed by others (Swift and Proctor 1977, Mlynski et al 2001 and others) applying noselike models in fluid dynamics experiments.

Nowadays, numerical models for airflow simulation play an increasingly important role. Numerical simulation is a method displaying a real environment (e.g., the human nose) within a computational model. Computational fluid dynamics (CFD) is a numerical simulation application to study various flow dynamics. The appropriate fluid flow physics are applied to the virtual nose model, resulting in a prediction of the fluid dynamics. It allows airflow patterns within the entire human nose to be displayed and analyzed and, if desired, simulations of the intranasal climate as well (Lindemann et al 2006, Keck and Lindemann 2010).

Inspiratory Airflow

The inspiratory airstream mainly follows the middle nasal passage (▶ Fig. 1.124 and ▶ Fig. 1.125). When passing through the external ostium, vestibule, and valve area, the air follows an upstream course that runs almost parallel to the nasal dorsum. After traversing the valve area, the airflow takes a more horizontal course. It hits the heads of the middle and inferior turbinates, enters the middle and, to a lesser extent, inferior nasal passages, and finally curves downward towards the choana and nasopharynx.

This relatively cranial course of inspiratory airflow is caused by the special anatomy of the external nose: the horizontal position of the nostril, the funnel shape of the vestibule, the position and configuration of the valve area, and the slope of the nasal dorsum. After passing the narrow valve area, the airflow becomes more turbulent for reasons already discussed (▶ Fig. 1.126). The relatively cranial course of the inspiratory airflow and the turbulence of the outer sheets of air promote longer and better contact between air and mucosa, as well as better contact with the olfactory area.

In laminar airflow, the direction of flow is parallel to the mucosal surface, with only the air film closest to the surface touching the nasal mucosa. The increased kinetic energy of the turbulent airflow allows an intensified contact between inhaled air and mucosa. The highest volume flows and flow velocities can be obtained in the center of the nasal cavity, followed by the inferior and middle meatus. The highest air pressure is detected at the heads of the inferior and middle turbinates. The areas surrounding the turbinates show vortices of low velocity with turbulence. Therefore, the turbinates seem to be responsible for the close contact between air and nasal wall (Lindemann et al. 2005).

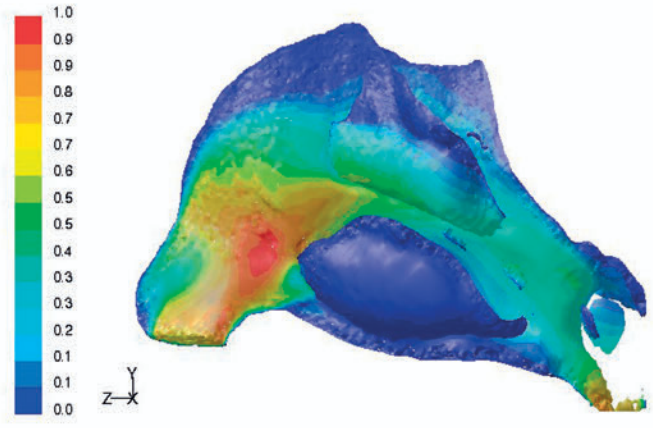

Fig. 1.124 Inspiratory contours of velocity magnitude (m/s) in a 3D nose model (lateral view).

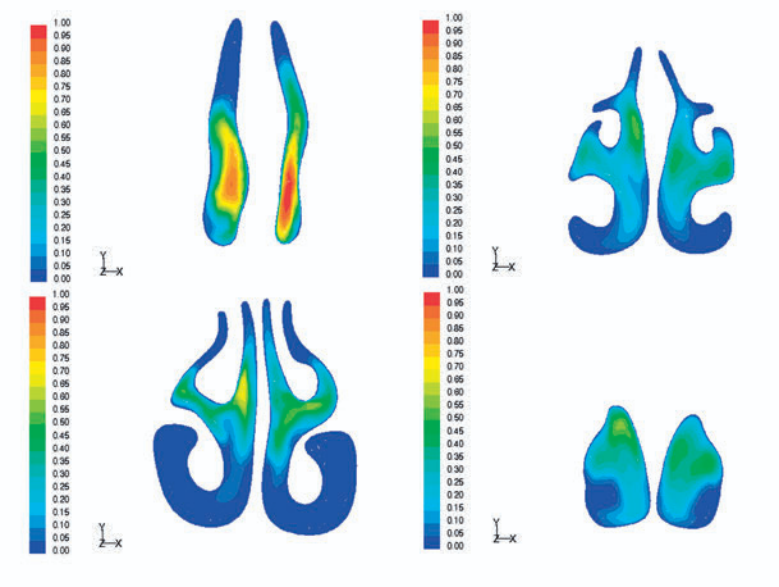

Fig. 1.125 Inspiratory contours of velocity magnitude (m/s) displayed on coronary cutting planes from anterior to posterior.

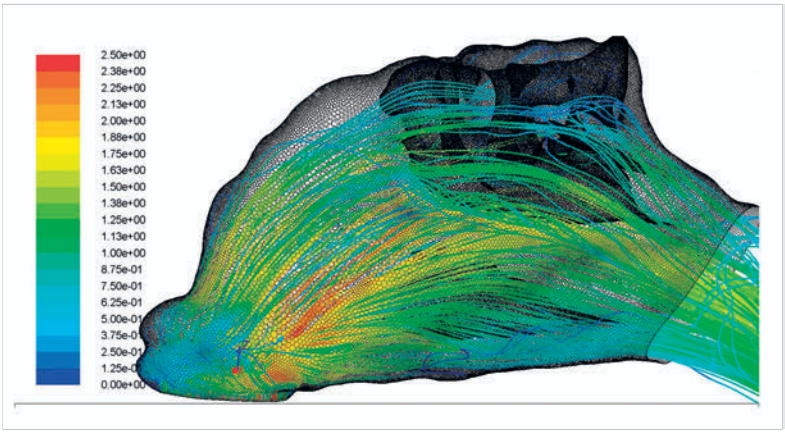

Fig. 1.126 Inspiratory airflow as path lines colored by velocity magnitude (m/s) (lateral view).

In addition to anatomical factors, the force of inspiratory breathing also plays an important role. The higher the inspiratory force, the higher the velocity of the airstream passing the narrow valve area. Consequently, the degree of turbulence of the air is increased, and the route taken by the air through the nasal cavity is more cranial. This is the case when we take a short, forced breath (sniff) to smell better.

Within the olfactory region, a slow, turbulent airflow with static vortices is prevalent, allowing intense contact between the inhaled air and the epithelium of the olfactory region (Lindemann et al. 2006).

The velocity of air during inspiration measures about 2 to 3 m/s at the nostril. It increases slightly in the vestibule, and then suddenly increases to 12 to 18 m/s at the valve area (▶ Fig. 1.127). Beyond the valve area, air velocity again decreases. In the nasal cavity it measures about 2 to 4 m/s.

Expiratory Airstream

The expiratory airstream takes a more caudal course through the nasal cavity, and mainly follows the inferior nasal passage. This is caused by the almost vertical position and relatively large diameter of the choana. The expiratory airflow is of the laminar type. The pressure differences from posterior to anterior are relatively small. Consequently, airflow velocity is low so Reynolds number is not attained. During expiration, the existing inspiratory turbulent airflow predominates in the posterior and middle nasal segment, and is bundled and transformed into a laminar one.

Physiology of the Valve Area

The nasal valve area constitutes the transition between the external and internal nose. It is a relatively narrow area measuring 50 to 70 mm^2. It is the major contributor

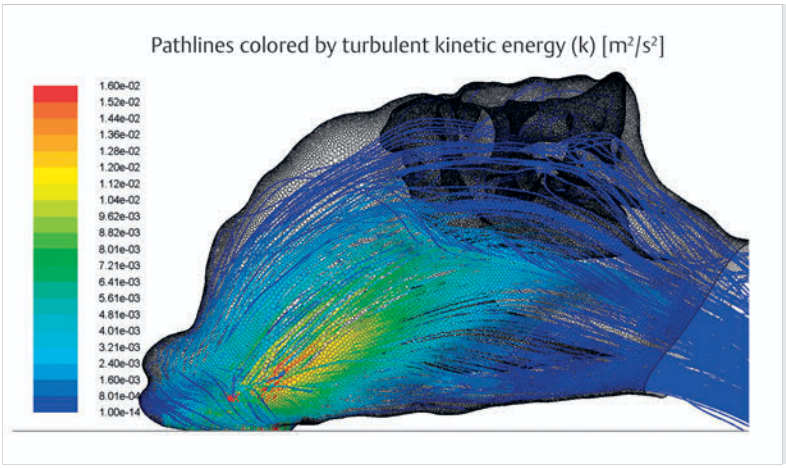

Fig. 1.127 Inspiratory airflow as path lines colored by turbulent kinetic energy (k) [m^2/s^2] (lateral view).

to nasal resistance, and consequently, a major regulator of nasal airflow. For the detailed anatomy of this area the reader is referred to ▶ Fig. 1.34 and ▶ Fig. 1.35.

Mink (1902, 1903, 1920) was the first to use the term *nasal valve*, in contrast to the 19th century anatomists (Zuckerkandl 1892), who spoke of the *ostium internum*, or *isthmus nasi*. Mink wrote that "the nose is provided with a valvular device... " and that "the valve rules the inflow of ambient air." Later authors, such as Van Dishoeck (1936, 1965) and Williams (1972), supported this concept. Bridger (1970) and Bridger and Proctor (1970) introduced the term *flow-limiting segment*, and compared the area with a Starling resistor (a semirigid tube with a collapsible segment). Haight and Cole (1983) located the resistive site "confined to a segment of a few millimeters at the junction of the compliant cartilaginous vestibule with the rigid bony cavity." They found nasal resistance increased from about 1.0 to 6.0 cm H_2O/L/s at a distance between 2.0 and 2.5 cm from the posterior margin of the nostril. They also found that the head of the inferior turbinate is an important contributor to this resistive area (▶ Fig. 1.128). The latter was confirmed by Jones et al (1988) in patients before and after "radical trimming" or "anterior trimming" of the inferior turbinates, and later again by Shaida and Kenyon (2000). Today, we prefer the term *valve area* to *valve* because it has become evident both in experiments and in clinical practice that the resistive area is a three-dimensional region and comprises several elements (Kaspenbauer and Kern 1987). Of these, the mobile caudal margin of the triangular cartilage and the more or less swollen head of the inferior turbinate are the most important. Other factors are the cartilaginous septum and the soft tissue covering of the floor of the piriform aperture. The valve proper is influenced by a number of anatomical and physiological factors, as we will discuss in the following text.

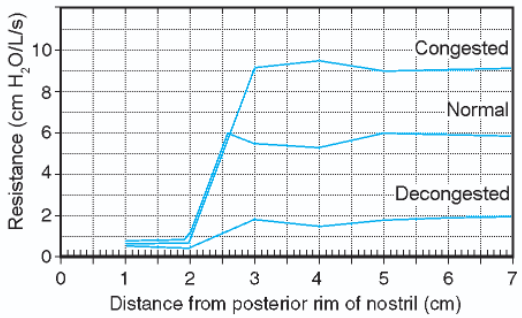

Fig. 1.128 Nasal resistance at different distances from the posterior rim of the nostril. In the normal nose, a strong increase in resistance occurs at 2.0 to 6.0 cm (i.e., the valve area). In the congested nose, this increase in resistance is considerably greater due to swelling of the head of the inferior turbinate. In the decongested nose, there is only a limited increase in nasal resistance. (Data from Haight and Cole 1983.)

Forces Acting on the Valve Area

The medial wall (septum) and the floor of the valve area are semirigid structures. The lateral wall, however, is somewhat flaccid and mobile. It moves inward during inspiration and outward during expiration. The magnitude of inward movement during inspiration depends on two factors (▶ Fig. 1.129):

1. The transvalvular pressure difference (difference between the pressure in the intranasal valve area and environmental air pressure), and
2. The compliance/rigidity of the lateral wall.

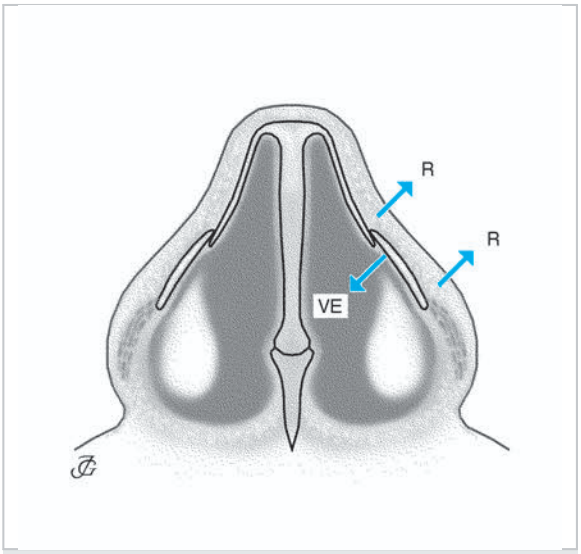

Fig. 1.129 Forces acting on the valve area during inspiration. VE = Venturi effect: the negative intravalvular pressure caused by increased air velocity; R = rigidity of the various anatomical components of the lateral nasal wall.

The magnitude of the transvalvular pressure difference, on the one hand, is determined by the force of inspiration and the cross-sectional area of the valve area. The narrower the valve area, the higher the velocity of inspired air and the greater the transnasal pressure difference (relative negative intravalvular pressure due to the so-called Venturi effect; see ▶ Fig. 10.54). The compliance of the lateral wall of the valve area, on the other hand, depends on four different factors. First factor is the dimension and thickness of the triangular cartilage and the presence or absence of returning of its lower margin. Second factor is the relationship between the lower margin of the triangular cartilage and the lobular cartilage (a greater degree of overlap will increase the rigidity of the lateral nasal wall). The third factor is the rigidity of the overlying connective tissue layers, skin, and the lateral soft-tissue area (hinge area) with its sesamoid cartilages. Finally, contraction of the nasal musculature (in particular the dilator, nasalis, and apicis nasi muscles) contributes to compliance of the lateral wall of the valve area (▶ Table 1.7).

Effect of Pathology at the Valve Area

The valve area is the most critical functional area of the nose. A limited stenosis at this area has great consequences for inspiratory breathing. Any narrowing will cause an increase in the transnasal pressure gradient, causing a greater degree of inward suctioning of the lateral wall and valvular collapse if its rigidity is not sufficient to counteract the negative pressure. It is well known that a minor septal deviation or convexity, or abnormal congestion of the head of the inferior turbinate, may be enough to induce this sequence of events. The same applies to pathological weakening of the lateral nasal wall after surgery or trauma.

Nasal Cycle

The human nose exhibits spontaneous changes in unilateral nasal resistance. When these changes are periodical and reciprocal we speak of a "nasal cycle" (Eccles 1997). The nasal cycle is caused by dilatation and constriction of the capacitance vessels in the nasal mucosa, in particular the inferior turbinates, in a rhythm of 3 to 5 hours. When the right nasal cavity is in a congested state, the left side is decongested, and vice versa. As a result, total nasal resistance and airflow will remain unchanged. This phenomenon is called the nasal cycle (▶ Fig. 1.130). Eccles pointed out that we are not dealing with a real cycle but a reciprocal (3:1) relationship of the resistance of both nasal cavities.

The nasal cycle was accidentally discovered by Kayser (1895) and has been an object of study ever since. The most important contributions have been from Lillie (1923), Heetderks (1927), Stocksted (1952, 1953), Keuning (1968), Masing (1969), Hasegawa and Kern (1978), and Eccles et al (1996, 1997, 2000).

The nasal cycle is controlled by the adrenergic system. It is regulated by a central modulating system located in the brainstem but is also influenced by local factors. The use of vasoconstrictive nose drops temporarily abolishes the mechanism. During nasal infection, the amplitude of the nasal cycle is increased.

In earlier studies, a nasal cycle was reported to be present in about 80% of adults with a normally functioning nose, as well as in children above the age of 3 to 5 years. Eccles et al (1997), applying more strict criteria, found a

Table 1.7 Activity of the nasal muscles during quiet breathing, after exercise, and during sniffing; the activity of the depressor septi muscle was not determined in this study (data from Bruintjes et al. 1996)

Muscle	Quiet breathing	After exercise	Sniffing
Procerus	—	±	±
Levator labii superioris	±	+	±
Pars transversa musculi nasalis	±	++	+
Pars alaris musculi nasalis	±	++	++
Dilator naris	±	++++	++
Apicis nasi	±	++++	++

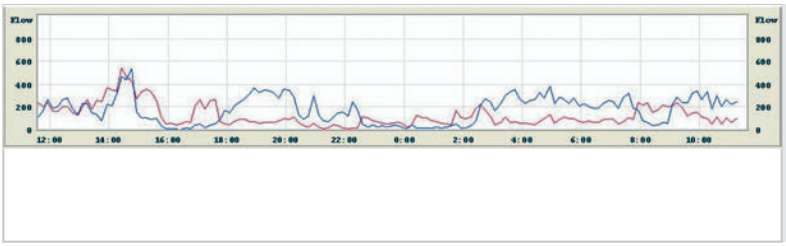

Fig. 1.130 Exemplary long-term rhinoflowmetry over 24 hours with normal nasal breathing, representing the nasal cycle. Red curve = right side; blue curve = left side; X-axis = time in hours; Y-axis = nasal respiratory volume at the maximum speed of inspiration in mL/s. (After data by Prof. G. Mlynski.)

real cycle to exist in only 20 to 40% of the adult population. It was also demonstrated in mammals.

The nasal cycle is present in all positions of the head and body. It has no effect on ciliary beat, but mucociliary transport may be influenced. The purpose of the nasal cycle is not well understood. A relation with homolateral pulmonary function has often been suggested but never proven. Eccles et al (1996) postulated that it serves as a defense. They hypothesize that in the congestion phase, the muscles around the venous sinusoids contract and squeeze out exudates. This would help cleanse the nose and enhance defense by releasing immunoglobulins and mediators.

Individuals with normal nasal function usually do not notice the alternating process of congestion and decongestion of the nasal mucosa. In pathological conditions, however, the nasal cycle may influence symptoms. Some patients complain of an alternating (left–right) breathing obstruction in the late (vasodilatory) phase of an infectious rhinitis, such as a common cold. Others notice a unilateral rhythmic obstruction on the side of a septal deviation.

A long-term assessment of nasal function and of the nasal cycle is still difficult to acquire. Therefore, the recently developed "long-term rhinoflowmetry" might present a new means to further investigate the nasal cycle.

Nasal Muscle Function

Electromyographical studies have shown that all nasal muscles are active at inspiration, in particular the nasalis, dilator naris, and apicis nasi muscles (e.g., van Dishoeck 1937, Bruintjes et al 1996). Contraction of these muscles widens the nostrils and increases rigidity of the lateral nasal wall, thus counteracting the risk of valvular collapse. Some muscle fibers insert at the cartilaginous structures of the lateral nasal wall; others end in the subcutaneous tissues.

All muscles attaching at the lateral nasal wall are "openers," not "compressors." Sea animals like the sea lion open their nares when surfacing by contracting the nasal muscles. When submerging, the muscles relax and the nostrils are closed.

In nasal surgery we try to preserve the nasal musculature as far as we can. Undermining of the dorsal skin should be as deep as possible—that is, immediately above the perichondrium and periosteum. In lobular surgery, the attachments of the muscle fibers to the cartilages (in particular those of the dilator muscle to the lateral crus) should not be cut.

1.4.3 Air Conditioning (Heating and Humidification)

The Mucosa as the Essential Organ of the Nose

Laymen think of the external nasal pyramid as the nose. Rhinosurgeons look at it as a three-dimensional complex structure that is made up of various anatomical–physiological components. They recognize the nasal mucosa as one component without always realizing that this mucosal lining of both nasal cavities performs most nasal functions. Intranasal air conditioning of the inhaled air and defense of the respiratory tract are the specific tasks of the mucosa, submucosa, and the parenchymal tissue of the turbinates. Therefore, the lateral wall of the nasal cavity is provided with a number of irregular protrusions—the turbinates—enlarging the functional nasal mucosa to a total surface area of about 150 cm^2.

Thus, the precious and irreplaceable nasal mucosa must be respected as much as possible in nasal surgery. Damage to the mucosa should strictly be avoided when performing septal and pyramid surgery, and even more importantly, when operating on the turbinates. Whenever possible, incisions in the mucosa should be avoided. Any approach is made through skin incisions. When a mucosal incision is unavoidable (e.g., draining a posterior hematoma), a horizontal incision is preferred over a vertical one, as a vertical mucosal scar may interrupt mucociliary transport.

One of the major functions of the nose, or rather the nasal mucosa, is to heat inspired air to almost body temperature (37°C) and humidify it to maximum saturation with water before arriving at the lower respiratory tract. For this purpose, the nasal organ is equipped with a large surface of mucosa with an extensive submucosal vascular network, a high density of secretory glands, and a rich nerve supply. The turbinate system was also developed to serve this task. The same applies, to some extent, to the nasal valve area, as it enhances the exchange of heat and water by changing nasal airflow from a laminar pattern to a more turbulent one within its narrow passage. This

emphasizes the very close relationship between intranasal air conditioning and airflow patterns (e.g., velocity, flow, vortices, path lines): the intranasal climate is mainly determined by airflow behavior within the nasal cavity. This is comparable with weather formation in nature.

The anterior nasal segment in particular, including the valve area and the turbinates, plays a crucial role in air conditioning. The space between the valve area and the head of the middle turbinate is the most effective part of the nasal cavity at heating and humidifying inhaled air.

When considering air conditioning, the expiration phase should not be neglected. Conditioning depends on *both* heating the air during inspiration *and* heat recovery during expiration. The crucial factor for water transfer from expired air to mucosa seems to be the temperature difference between the mucosal surface and the respiratory air.

The nasal surgeon should be aware of these physiological mechanisms and try to restore them when they are compromised by pathology or previous surgery.

Heating and Humidification

Several in vivo investigations revealed that an air temperature of about 31 to 34°C and a relative humidity of about 90 to 95% after inspiration could be observed within the nasopharynx (Keck and Lindemann 2010). However, nasal heating and humidification are not complete at this level. Further warming and humidification up to 37°C and 100% relative humidity occurs to a minor extent within the lower airways.

Due to the fact that most of the conditioning takes place within the anterior nasal segment, short-term exposure to cold, dry air or warm, humid air does not impair nasal air conditioning (Keck et al 2001). The nose has a large reserve for heating and humidification. The temperature difference between the mucosal surface and the respiratory air is a crucial prerequisite for heat exchange between the two. The temperature of the nasal mucosa depends on the phase of the respiratory cycle and the exact intranasal detection site. The mean mucosal temperature during respiration ranges from 30°C at the end of inspiration to 34°C at the end of expiration (Lindemann et al 2002). This temperature gradient between the mucosal surface and inspired as well as expired air is essential for effective heat and water transfer.

During inspiration, the warmer nasal wall heats the cooler air; during expiration, the cooler wall cools down the warmer air. In addition, water is regained from saturated and warmed expired air in the cooler mucosa. Thereby, water is preserved for humidification of air during the following inspiration. Thus, the loss of heat and water is reduced (Keck and Lindemann 2010).

There is also a close relationship between nasal airflow patterns and nasal mucosal temperature (Lindemann et al 2006). In regions of turbulent airflow, temperature changes are more pronounced than in regions of laminar airflow. This fact again confirms the close relation between airflow and intranasal air conditioning.

Nasal surgeons should not forget that any surgical intervention at the head of the inferior turbinate alters the valve area, and may lead to considerably disturbed air conditioning.

Mucosal Temperature and Perception of Nasal Patency

Difficulty in nasal breathing is a common complaint. At present, we mainly rely on a good clinical examination to identify the underlying problem causing nasal obstruction (Scadding et al 2011). For medicolegal or insurance purposes and for clinical trials, more objective means of measuring nasal patency and/or flow are often warranted, but data often correlate poorly with the subjective feeling of nasal obstruction. The feeling of nasal obstruction may have an underlying anatomical, mucosal, or physical etiology. Anatomical and mucosal causes of nasal obstruction are well known, but physical factors related to nasal obstruction are less studied. The existence of cold receptors in the nasal cavity has been verified. The subjective perception of nasal flow actually seems to be related to the activation of these receptors. For example, L-menthol causes a subjective improvement of nasal patency due to vapor action on the sensory nerve endings of the nasal mucosa, without objectively increasing measured nasal patency values (Eccles, Lindemann et al 2008). Additionally, there is a negative correlation between mucosal temperature and rhinometrical airflow volumes: high nasal flow is associated with low mucosal temperature (Lindemann et al. 2009).

The physiological perception of nasal airflow could be based on the cooling of the nasal mucosal surface by the air jet. This supports the hypothesis that the presence of nasal thermoreceptors plays an important role in the perception of nasal patency. These findings emphasize the fact that adequate heat and water exchange seems to be necessary for the perception of airflow and the feeling of a free nose.

1.4.4 Defense

The nose is provided with a number of different mechanisms to protect the airways: mechanical, humoral, and cellular defense.

Mechanical defense. The first line of mechanical defense consists of the vibrissae at the nostril and the vestibule. They protect the airways against incoming larger objects such as insects, although only to a limited extent. The second line of mechanical defense is the mucous blanket covering the mucosal membranes, in which smaller particles are entrapped and subsequently transported to the nasopharynx by the coordinated movements of the MCT. The third barrier is the epithelial lining, being a physical barrier between the lumen and the nasal tissue.

Humoral defense is provided by the production of immunoglobulins (IgA and IgG) and various enzymes at the level of the nasal mucosa. The role of humoral defense mechanisms in airway homeostasis becomes apparent in patients with dysfunction of Ig production, resulting in recurrent respiratory tract infections.

Cellular defense is mediated by a large variety of cells that may be recruited to help counteract the effects of allergens, viruses, bacteria, molds, etc. Eosinophils are the hallmark of allergic rhinitis, but are also attracted to the nasal mucosa in some forms of nonallergic rhinitis and rhinosinusitis with or without nasal polyps.

Filtering of the Air

Nasal cleansing of particles involves several processes. *Filtration* is removal of particles from respiratory air. *Deposition* is removal of particles by sedimentation on the nasal mucosa. *Retention* is the capture of mainly gaseous particles of the air. *Clearance* means removal of deposited particles on the mucosa by ciliary activity.

Nasal deposition depends on the attributes of the inspired particles (hygroscopic or hydrophobic, size, aerodynamic diameter, surface, density, and other chemical variables). Particles with a diameter between 0.4 and 3.0 µm mostly pass the nasal airways and are mainly deposited in the bronchial airways. Particles with a diameter smaller than 0.4 µm and bigger than 10 µm are mainly filtered in the nose (e.g. Keck et al 2002).

As a result of gravity and turbulence of the inspired air, some of the particles that are present in inhaled air will be deposited on the mucous layer covering the mucosal membranes. Larger and heavier particles will be deposited sooner and thus more anteriorly. They are entrapped in the upper layer of the mucous blanket. When insoluble, they are transported with a relatively high speed (0.5 to 2.0 cm/min) towards the pharynx by ciliary movement, and swallowed. To this end, the mucous layer consists of two sheets: an upper, more viscous and sticky gel in which the foreign particles are entrapped; and a lower liquid layer that allows ciliary movement. Soluble particles may dissolve in the deeper periciliary mucous layer and affect the mucosa.

Similar to climatization, particle filtration depends on respiratory parameters such as breathing frequency and tidal volume.

Cilia

Movements of the cilia covering the columnar cells of the mucosa are the driving force behind transportation of mucus towards the nasopharynx (MCT). A normal ciliary beat consists of an effective and a recovery stroke. During the effective stroke, the cilium is stretched and reaches the gel layer with its tip. This leads to propulsion of the upper viscous layer of mucus. In the recovery stroke, the cilium curves and bends backward to its rest position in a plane parallel to the epithelial surface. It then enters a short rest phase (▶ Fig. 1.131). The effective stroke of various neighboring cilia and cells is coordinated in a synergistic way. The mechanism behind this ciliary coordination or metachron is not yet elucidated.

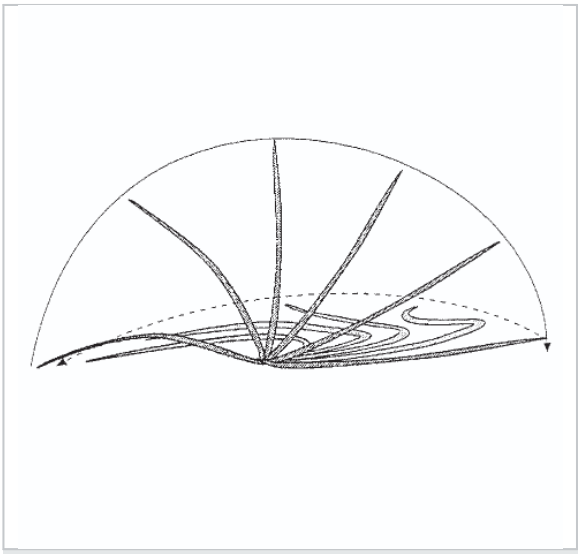

Fig. 1.131 Effective and recovery strokes of the ciliary beat and their different planes. (From Schuil 1994.)

Under normal circumstances, ciliary beat frequency (CBF) is 8 to 11 Hz at the normal nasal mucosa temperature of 33 to 34°C. An increase in CBF leads, within a certain range, to an enhancement of MCT (Boek et al 1999). CBF is sensitive to a large number of influences, however. Desiccation of the mucosa leads to immediate abolishment of ciliary activity and loss of cilia. Temperature increase produces, within a certain range, an increase in CBF. Changes in pH (between 6.5 and 7.5) and osmolarity (between 300 and 400 mol) have no effect (Ingels et al 1991).

Various mediators and neuropeptides that play a role in nasal (patho)physiology have been found to inhibit or enhance ciliary activity (Schuil et al 1994). Our present knowledge is summarized in ▶ Table 1.8. Several pharmaceutical products applied to the nose for diagnosis and treatment have also been shown to affect ciliary beat (▶ Table 1.9). In particular, reference is made to the preservative benzalkonium chloride, a frequent ingredient in nasal sprays.

Mucociliary Transport (MCT)

The superficial viscous mucous layer covering the nasal mucosa is propelled at a speed of 0.5 to 2.0 cm/min in a dorsal direction by the to-and-fro movements of the cilia. These cilia symbolically move as ears of corn in the wind. MCT velocity may vary considerably. It may be influenced by various factors such as the quality of the mucous

Table 1.8 Effect of the most important mediators and neuropeptides on CBF

	No effect	Inhibition	Enhancement
Histamine	+ (in vitro)		+ (in vivo)
Leukotriene C4	+ (in vitro)		
Prostaglandin D2	+ (in vitro)		
Prostaglandin E2		+ (in vitro)	
Substance P	+ (in vitro)		+ (in vivo)
CGRP			+ (in vitro)
Nitric oxide			+ (in vitro)
Interleukin 13		+ (in vitro)	

Abbreviations: CBF, ciliary beat frequency; CGRP, calcitonin gene–related peptide.

layers, CBF and ciliary coordination, and turbulence of the inspired air.

For an ideal functioning of the MCT, a temperature of 37°C and a relative humidity of 100% is required. If there is insufficient heat and humidity, the ciliary cells stop functioning after a short time. The bacterial colonization is facilitated under these conditions and infections may result. Therefore, MCT and intranasal air conditioning are also closely related.

It is scientifically presumed that cilia have a sensory function in addition to their purely mechanical function. If applied, for example, bitter substances such as nicotine or quinine have contact with ciliary cells, consecutively the calcium concentration within the cells increases. The cells are activated and the CBF is increased in order to remove pollutants from the nasal mucosa.

The methods used to examine cilia and to measure CBF and MCT in clinical practice are discussed on page 121.

The nasal surgeon should keep in mind the importance of this defense mechanism. He must try to restore it as far as possible, a normal breathing pattern should be restored and the nasal mucosa preserved.

Humoral Defense

The nasal mucosa is provided with extensive and complicated mechanisms of humoral defense that have not yet been elucidated.

Immunoglobulins. IgA and, to a lesser extent, IgG are considered to play a major role in nasal defense. Secretory IgA concentration in nasal mucus is very high at 0.5 to 2.2 g/L—higher than in saliva or intestinal secretions.

Table 1.9 Effect on CBF of some pharmaceuticals topically used in the nose (after data by Ingels, Boek, and Schuil)

Pharmaceutical agent	No effect	Inhibition	Enhancement
Topical anesthetics			
Cocaine 3%		++	
Cocaine 7%		+++ (stasis)	
Lidocaine 1%		++	
Lidocaine 4%		+++ (stasis)	
Vasoconstrictors			
Xylometazoline 0.1%		+	
Oxymetazoline 0.05%		+	
Preservatives			
Benzalkonium chloride		+	
Saline 0.9%		+	
Saline 7.0%		+++ (stasis)	
Antifungals			
Amphotericin B		+++	
Itraconazole		+++	
Other			
Corticosteroids		+	
Carbachol			+
Salbutamol			+

Abbreviations: CBF, ciliary beat frequency.

Its concentration is not related to serum IgA level. IgG seems to play a smaller role. Its concentration only amounts to 0.2 g/L. IgM concentration in nasal secretions is very low. Immunoglobulins are produced in the nasal mucosa by plasma cells and B lymphocytes.

Histamine is the most potent compound, which is released by mast cells and basophils during the immediate phase of the (IgE-mediated) allergic reaction. It is responsible for the most important symptoms in allergic rhinitis: nasal congestion, secretion, and sneezing.

Leukotrienes (LTB4, LTC4) play a similar role to histamine during immediate allergic reactions. They are also involved in the continuing "inflammatory" response.

Prostaglandins (PGD2, PGE2) are another group of mediators produced by mast cells during the allergic response. Their role is not yet elucidated.

Interleukins are a group of cytokines produced by cells in the nasal mucosa and are part of the inflammatory spectrum seen in rhinitis and rhinosinusitis. Interleukins are released upon activation of lymphocytes, mast cells, neutrophils, eosinophils, epithelial cells, and fibroblasts present in nasal tissue. Several major interleukins have been identified, and the most important ones at present seem to be IL-4, Il-5, IL-8, IL-12, IL-13, and IL 17. The wide variety of functions of this growing family of cytokines are still under exploration, but several have redundant and heterogeneous effects on epithelial cells, fibroblasts, mast cells, endothelial cells, and sensory nerves residing in the nasal mucosa.

Activation of endothelial and epithelial cells leads to upregulation of the expression of *adhesion molecules*, which are responsible for the influx of inflammatory cells at the site of immune stimulation by microbial or environmental triggers. So far, three main groups of adhesion molecules have been described: selectins, integrins, and the immunoglobulin superfamily.

Cytokines may have proinflammatory potential, but some, such as IL-10 and transforming growth factor-beta (TGF-B), have anti-inflammatory actions. These so-called anti-inflammatory cytokines are considered to protect the upper airways against inflammatory damage.

Cellular Defense

The nasal mucosa, submucosa, and secretions contain numerous types of cells that play a role in the different types of respiratory mucosal defense.

Dendritic cells reside in the nasal mucosa. They capture pathogens and allergens, migrate to the draining lymph nodes, and present parts of the pathogens and allergens to T and B lymphocytes. In this way, an adequate immune response is initiated, on both a cellular and a humoral level.

Eosinophils are the most well known. Their number in the mucosa, submucosa, and in nasal secretions is specifically increased in allergic rhinitis (IgE-mediated allergy). Their number is related to the severity of symptoms. Their granules contain various proteins, toxins, and enzymes.

Mast cells were found long ago to play a role. They are present in the submucosa and, in allergic reactions, migrate to the mucosal surface, where they degranulate and release a great number of substances, such as histamine, platelet activating factor (PAF), different enzymes, and cytokines. Some of these enzymes induce the release of prostaglandins (PGD2, PGE2) and leukotrienes (LTB4, LTC4).

Basophils are also present in the submucosa, but they increase in number and migrate to the mucosal surface in allergic challenge. They release histamine and LTC4, among others.

Plasma cells are recruited in various conditions. They play a dominant role in the production of immunoglobulins.

T lymphocytes are scattered in high numbers in allergic conditions. CD4 (helper cells) strongly prevail over CD8 (suppressor) cells. When activated, they produce various cytokines.

B lymphocytes are among the producers of IgE.

Nasal Reflexes

The basic functional role of the nose in human physiology is also illustrated by the large number of nasal reflexes. Some of them are pure defensive reflexes; others are signs of the complex relationship between the nose and other physiological systems.

Naso–Nasal Reflex (Sneezing)

The sneezing reflex is the most important sensory (trigeminal) defensive reflex. It may occur as a reaction to a wide range of physical and chemical nasal stimuli. Even bright light can cause sneezing (photic sneezing), which can be observed in 17 to 35% of people. Its causes are not yet clarified. However, an autosomal dominant inheritance, the so-called ACHOO syndrome (Autosomal Dominant Compelling Helio-Ophthalmic Outburst of Sneezing [Collie et al 1978]) is assumed. The most common theory is an abnormally close course of the optic nerve to the trigeminal nerve. In cases of sudden brightness, action potentials are conducted along the optic nerve, stimulating the trigeminal nerve as well. This is perceived cerebrally as an irritation of the nasal mucosa, finding its expression as a sneeze.

Sneezing occurs in three phases. In the first phase, air is deeply inhaled. The breath is held briefly (second phase), and then the expiratory muscles suddenly contract (the third phase). The air is strongly exhaled against a closed glottis. The larynx and pharynx are then opened and a short explosive expiration follows through the nose (and mouth). Simultaneously, via local and central parasympathetic efferent pathways, nasal vasodilatation and secretion are induced. The airflow velocity reaches over 45 m/s. This sneezing "reflex" is not a real reflex as it is neurally too complex and can be deliberately influenced. It is eliminated by local and general anesthesia.

Naso(Laryngo)bronchial Reflex (Nasopulmonary Reflex)

A second reflex with a defensive nature is the nasopulmonary reflex. This reflex has been studied for a long time and its significance is still a matter of dispute. It is an ipsilateral reflex, with the sensory trigeminal nerve endings of the nasal mucosa as its afferent, and vagal fibers as its efferent, pathway. Nasal stimulation, for example cold air, may induce a reduction in breathing—even apnea—and laryngeal and bronchial constriction. This naso(laryngo)bronchial reflex may play an important role in breathing distress, especially in the elderly. Several studies have demonstrated that nasal obstruction or nasopharyngeal packing may cause a decrease in arterial oxygen saturation and an increase in blood carbon dioxide. The nasal surgeon and the ENT doctor applying a tamponade (particularly a Bellocq type) for epistaxis should be aware of this phenomenon.

In allergic rhinitis and in sinus disease, (sino)nasal inflammation and bronchial pathology interact in various ways. Neural pathways are clearly involved, with mediators like substance P being upregulated in bronchi after nasal stimulation. In addition, the systemic circulation plays a role in this nasobronchial interaction by transporting cytokines, such as IL-5 released in the upper airways to the blood, resulting in enhanced bone marrow synthesis of inflammatory cells and upregulation of adhesion molecules on bronchial endothelial cells. In addition, allergens that are deposited in the nasal mucosa enter the submucosal area and blood vessels, leading to activation of systemic basophils. Clinically, these immunologic phenomena translate into a close interaction between (sino) nasal inflammation and bronchial pathology, both in allergy as well as in rhinosinusitis. Conversely, bronchial asthma or chronic obstructive pulmonary disease (COPD) have a negative impact on (sino)nasal disease, with more severe inflammation and symptoms and a worse outcome after functional endoscopic sinus surgery (FESS) (Hellings et al 2010).

Corporonasal Reflex (Diving Reflex)

The diving reflex is a protective mechanism in all lung-breathing creatures when immersed in water. The brain receives information that the airways are under water. Stimulation of the parasympathetic nervous system due to immersion of, for example, the face, chest, feet, and back in cold water leads to reduced breathing (even apnea), bradycardia, and centralization of the bloodstream ("blood shift"). Hereby, the oxygen consumption of vital organs is reduced.

Apart from the above-described reflexes, several other nasal reflexes have been described and studied. These reflexes are clinically less important. Therefore, they are only listed in brief.

Nasocardiac Reflex (Cranial Nerve V–Cranial Nerve X)

Strong stimulation of the nasal mucosa produces bradycardia and a reduction of cardiac output with lowering of the blood pressure.

Nasovascular Reflex

Nasal stimulation causes peripheral vasoconstriction.

Genitonasal Reflex

Sexual arousal and orgasm cause swelling of the nasal mucosa, particularly of the turbinates.

Gastronasal Reflex (Cranial Nerve X–Parasympathicus)

Strong gastric stimulation by irritation (e.g., alcohol, coffee) or gastritis may cause nasal secretion and vasodilatation on the homolateral (left) side of the nose.

1.4.5 Speech

The nose and the paranasal cavities are an essential part of the speech-production apparatus. The nasal cavity is one of the resonators that play a role in production of some vowels and several consonants. Typical examples are "m" and "n," the so-called nasal consonants.

The amount of nasal resonance is called "nasalance." Previously, it was common to speak of "rhinolalia clausa" when nasal resonance was decreased, and of "rhinolalia aperta" when nasal resonance was present in consonants requiring closure of the nasopharynx by the soft palate.

Many rhinological patients suffer from decreased nasalance because of nasal obstruction due to mucosal swelling, polyposis, or septal deformity. Surgery may have a considerable effect on their speech and singing. Professional (and amateur) singers may benefit considerably from functional reconstructive nasal surgery.

1.4.6 Facial Beauty and Facial Expression

The nose plays a dominant role in concepts of facial beauty and expression. In almost all cultures, we find indications of the dominant role that the shape of the nose plays in life. A nose perceived as beautiful enhances facial beauty and helps make a person attractive. A deformed or damaged nose may be perceived as ugly.

Beauty

Different cultures have developed different concepts of nasal beauty. We find evidence of this in the special "rules" that were conceived for describing ideal body proportions, and from works of art.

When discussing beauty, it is of utmost importance to take into consideration the many ethnic differences in the shape of the face and nose. The faces and noses of blacks and Asians are very different from those of the Caucasians. The rhinoplasty surgeon should be aware of this and should attempt to preserve some ethnic character when performing an operation.

Aversion to One's Own Nose

Many patients visit the nasal surgeon because they dislike their nose. In many cases their problem is understandable: a deviated pyramid, a large hump, a severe saddle, etc. Mostly, their complaints are not only cosmetic; function is impaired too. Where there is no real "deformity" but a minor "abnormality" or nothing more than a "variation," the surgeon should be warned. A careful analysis of the "problem," sometimes including a psychological analysis, is then required. Nothing is more disappointing, both for the patient and the doctor, than a patient who is dissatisfied with a "good" result. According to recent studies, a significant number of patients with aesthetic nasal complaints have symptoms of a body dysmorphic disorder (BDD).